Physiogenex
THE METABOLIC DISEASES EXPERTS

Diet-induced NASH (DIN) mouse model associated with metabolic syndrome

- ✓ Unique diet-induced mouse models of non-alcoholic steatohepatitis (NASH)
- ✓ Associated with obesity and insulin resistance like in a human NAFLD physiopathological context

Key benefits

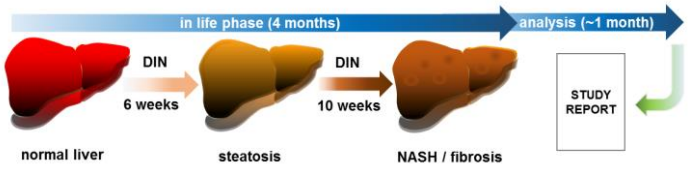
Unique proprietary diet-induced animal models that enables pharmacological studies targeting NASH and fibrosis, in a human-like context including obesity and insulin resistance.

The diet–induced (DIN) NASH mouse provides:

- A pharmacologically validated model to study NASH and fibrosis, associated with metabolic syndrome
- Allows to study mechanisms involved in NAFLD progression
- Predictive model: similar to human situation where the diet plays a major role in the development of NAFLD

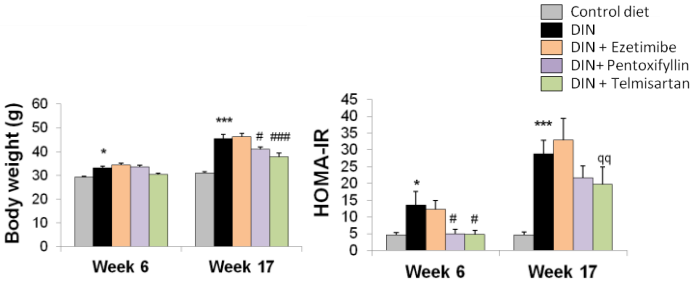
ANIMAL MODEL

- Background strain/gender: C57BL/6J mice, male
- In house "Diet-Induced NASH" (DIN™): High Fat +cholesterol + fructose in drinking water
- Reference compounds: ezetimibe, pentoxifyllin and telmisartan
- Experimental design:

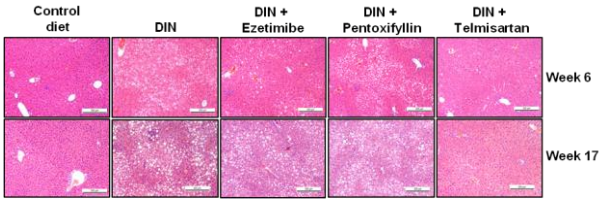


PATHOPHYSIOLOGICAL FEATURES

DIET INDUCED OBESITY AND INSULIN RESISTANCE

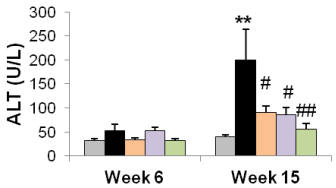


LIVER STEATOSIS (H&E staining)



LIVER INJURIES

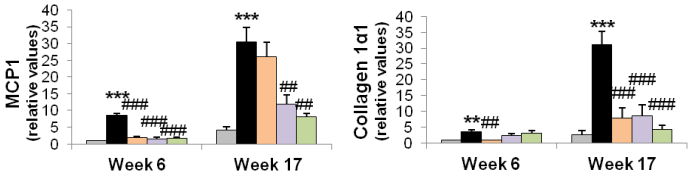
- *Plasma biomarker*



*p<0.05, **p<0.01, ***p<0.001 *vs.* control diet, #p<0.05 #p<0.01 ###p<0.001 *vs.* DIN, qq<0.01 *vs.* DIN without ezetimibe group

LIVER GENE EXPRESSION

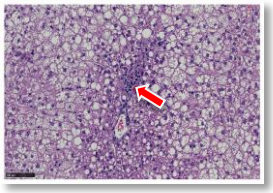
Inflammation (qPCR) *Fibrosis (qPCR)*



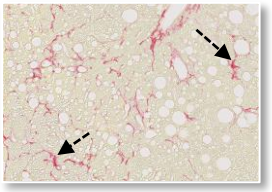
NAS SCORING AT 16 WEEKS OF DIN

- *Histology*

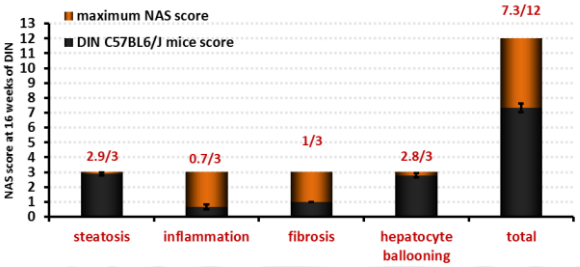
mononuclear cell infiltration
Fibrosis



H&E staining



Sirius Red Staining



END-POINTS

- Anatomopathology (histology, immunohistology, NAS score)
- Plasma and liver biomarkers:
  - lipids, inflammation
  - liver enzymes
  - liver gene (qPCR) and protein expression : standard biomarkers and others on request

REFERENCES

Dubuquoy C et *al*. Effects of pharmacological compounds on *in vivo* models of NAFLD associated to metabolic syndrome. SFD, 2014.

Sulpice T. et *al*. Prevention of liver damages by targeting different physiological mechanisms in a new murine NASH model associated with metabolic syndrome. World Diabetes Congress, 2013.

Prologue Biotech – 516, rue Pierre et Marie Curie
31670 Labège - FRANCE

Phone : +33 561 287 040
Fax : +33 561 287 043

www.physiogenex.com
business@physiogenex.com

Member of cardio metabolic